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Background piece

SUMMARY OF SCIENTIFIC TESTIMONY
BEFORE THE SENATE COMMITTEE ON COMMERCE
March 26 - April 2, 1965

Testifying before Senate hearings on proposed bills which would require health warnings to be placed on cigarette packages and/or in cigarette advertising, 37 scientists, physicians and other experts questioned the conclusions drawn in "Smoking and Health," the 1964 Report of the Advisory Committee to the Surgeon General.

The Report says (pp. 26-7) the SGAC drew its conclusions from what it called converging lines of evidence in three general categories: (I) Statistical (population) studies, (II) Animal experiments, and (III) "Clinical and autopsy" studies.

This is a summary of the principal points made by scientific witnesses in questioning the SGAC Report. While many of the witnesses touched on all three areas, each usually discussed his own specialty. This summary indicates in parenthesis the names of the witnesses who made the point in question, with appropriate page references to their testimony.

(I) Statistical (population) studies

Noting that the Report chiefly relies upon seven statistical surveys, the statistical experts among the witnesses found these studies deficient in both materials and methods. Their principal criticisms were:

(1) These seven population groups are not representative of the U.S. population or any other known population, as the Advisory Committee itself acknowledged in its Report (p. 94). (Actually, the

death rates for cigarette smokers are lower than the death rates for U. S. males as a whole, in some of the studies cited). (Donnahoe, p. 11; Sterling, p. 14; Huff, pp. 4-5). Also, the death rates in several studies are not only lower than that of U. S. white males as a whole, but also differ from study to study. (Sterling, p. 14).

(2) There were high percentages of non-response in some of the surveys, and haphazard selection of respondents in others. (Donnahoe, p. 12; Huff, pp. 4-5; Brownlee, p. 3; Sterling, pp. 12-13) Both these factors may have biased the samples, and at best make the study populations impossible to define. (Sterling, p. 13)

(3) The statistical association between smoking and disease is not specific -- that is, smoking is associated with higher death rates for almost all 25 of the diseases the SGAC studied, not just lung cancer. Nevertheless, the Committee concluded that the data was not sufficient to support a causal connection for nearly all of these diseases. (Donnahoe, p. 8)

(4) Lung cancer occurs in persons who have never smoked. (Carr, p. 4. A majority of the witnesses made this point, as well.)

(5) The samples used were sometimes not big enough. For example, the Report's conclusion that cigarette smoking causes cancer of the larynx was based on four studies in which the number of cases of the disease were respectively 17, 14, 7 and 3. (Huff, pp. 6-7)

(6) Half of the reported increase in cancer, including lung cancer, as the SGAC Report shows on p. 128, is due to population growth and

aging. An unknown part of the other half of the increase may be due to improved diagnostic methods and changes in reporting. (Huff, p. 13)

(7) Statistical association alone cannot prove cause and effect, as the Report states on p. 20. (Huff, p. 18; Donnahoe, p. 18)

(8) Where the evidence did not fit the hypothesis the Committee was exploring -- smoking as the cause of various diseases -- the SGAC either explained it away or denied its existence. (Brownlee, p. 12; Saiger, pp. 3-4; Sterling, p. 14)

(9) The SGAC admitted various possible sources of error in the statistical data, and then guessed at the degree to which these errors might have affected the data. (Donnahoe, p. 16)

(10) The apparent statistical correlation between cigarette smoking and various diseases may be without causal significance since smoking and disease are both correlated with other factors, such as psychological and physiological differences between people. (Donnahoe, p. 17; Russek, p. 4; Brownlee, pp. 5-11; Sachs, p. 5; Wolffe, p. 3; Saiger, pp. 9-15); and smoking and susceptibility to disease may both be expressions of a common genetic trait. (Brownlee, pp. 5-11)

(11) The peak age of lung cancer incidence is 50-65 years of age, regardless of how much or how long a subject smokes, or whether he smokes. This fact tends to dispute statistical studies indicating that lung cancer incidence itself is related to amount smoked, or duration of smoking. (Mayer, p. 4; Clerf, p. 6; Macdonald, p. 10; Garland, pp. 4-5; Langston, pp. 5-6; Barrett, pp. 10-11).

(12) Cancer of the lung is more common in Holland, Switzerland, Finland and Great Britain than it is in the United States, yet all four countries consume fewer cigarettes per capita than we do. (Donnahoe, p. 5)

(13) Cancer of the trachea or windpipe is a very rare disease, although the windpipe receives as great or greater exposure to tobacco smoke than the part of the lung where most lung cancers are said to develop. (Helwig, p. 3; Barrett, pp. 7-8; Garland, p. 3; Clerf, p. 3; Ogura et al., p. 2; Moran, p. 4)

(14) Other factors, such as alcohol and inadequate nutrition, may play a significant role in larynx cancer. In some studies, alcohol is more strongly associated with larynx cancer than is tobacco use. (Ogura et al., pp. 3-4)

(15) The technique of diagnosis of laryngeal cancer has remained essentially unchanged over the past three decades, while great advances have been made in diagnosing lung cancer over the same period. This accounts at least in part for the fact that the reported incidence of laryngeal cancer has remained static, while the reported incidence of lung cancer has increased -- although the SGAC Report attributes both diseases to the increase in cigarette smoking. (Clerf, p. 6)

(16) There has been no increase in laryngeal cancer despite increased cigarette consumption. (Ogura et al, p. 2; Clerf, p. 3)

(17) Lung cancer occurs much more frequently among men than women, and in the past 40 years the gap has widened substantially, although smoking among women has been growing rapidly for the last 40 years.

(Moran, p. 4; Mayer, p. 5; Rosenblatt, pp. 4-5) This is also the case with larynx cancer. (Ogura et al, p. 3)

(18) The increase in reported lung cancer cases began prior to the popularity of cigarette smoking. (Rosenblatt, p. 3)

(19) There has been a progressive decline in the rate of increase of lung cancer, during a period in which cigarette consumption increased 200 times. (Rosenblatt, pp. 3-4; Barrett, p. 3; Greer, p. 6)

(II) Animal Experiments

(20) Experiments in which animals were forced to inhale tobacco smoke have repeatedly failed to produce lung cancer. (Carr, p. 1; Sterling, p. 2; Report, p. 33) Other inhaled substances have produced lung cancer in animals. (See point 24)

(21) There is no experimental evidence to support the SGAC conclusion that cigarette smoking causes larynx cancer. (Ogura et al, p. 6)

(22) Once you go beyond the statistical studies, there is little relevant experimental or clinical evidence to link cigarettes to lung cancer. While some skin cancers have been produced by applying heavy doses of tobacco condensates ("tar") to the skin of susceptible animals, similar cancers can be produced by painting animals with a variety of substances, including some common foods. (Moran, p. 5)

(23) Skin-painting experiments have shown pipe-tobacco smoke condensates are more active in producing skin tumors in mice than are cigarette-smoke condensates, though the statistics suggest that pipe smoking is innocuous for humans. Such results indicate that skin-painting experiments are irrelevant to the human cancer problem. (Hockett, p. 18)

(24) Animal lung cancers have been produced through the application of viruses, ozonized gasoline, bacterial toxins and dilute acids but not with tobacco smoke. (Sprunt, pp. 6-7; Hockett, p. 21; Moran, p. 5)

(III) "Clinical and Autopsy" Studies

(25) The tissue changes in smokers' lungs reported (by Auerbach and some others) are also common in the lungs of non-smokers, including children. (Sprunt, p. 5; Ober, pp. 6-7,; Moran, p. 6; Carnes, p. 8)

(26) The changes are associated with a wide variety of conditions, infections in particular. (Sprunt, p. 5; Ober, pp. 6-7; Moran, p. 6)

(27) The changes are reversible -- under certain conditions, they disappear. (Moran, p. 6; Sprunt, pp. 5-6)

(28) There is no evidence that these changes lead to cancer. Long term experiments with animals show that similar changes do not progress to cancer. (Ober, pp. 6-7; Sprunt, pp. 4-5; Moran, supplemental testimony, p. 3)

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(29) Most lung cancers appear to start in a small bronchi and move later to the larger bronchi, although the small bronchi have the least exposure to tobacco smoke. (Mayer, p. 5; Burford, p. 2)

(30) Among the possible factors involved in lung cancer needing further study are viruses, industrial or occupational exposure, atmospheric pollution, chronic inflammation and scarring of the lungs such as occurs after influenza or repeated infections, and psychological and constitutional differences among people, as well as smoking. (Burford, p. 4; Mayer, pp. 6-7; Greer, p. 6; Brownlee, pp. 5-11)

(31) There is little beyond statistical evidence to suggest a causal relationship between smoking and heart disease. Smoking may

be nothing more than a symptom of an underlying cause, such as stress. (Wilens, p. 1; Kaplan, p. 5; Wolffe, pp. 3-4; Russek, p. 3)

(32) Excessive smoking, like overeating and failure to obtain adequate exercise, is often an outward manifestation of psychological stress. (Russek, p. 3)

(33) Studies suggest that emotional stress is far more important than tobacco in the development of heart attacks. (Russek, p. 1; Wolffe, p. 4)

(34) A study of 25,000 professional men found that those who reported that they had never smoked showed a greater frequency of heart attacks than those who had stopped smoking. (Russek, p. 4)

(35) An autopsy study indicates that heart ~~attacks are~~ damage is no more frequent among smokers than non-smokers. (Wilens, pp. 3-4)

(36) Current acceptance of the cigarette hypothesis may deter further research into possible causes. (Carr, p. 9; Mayer, p. 7; Greer, p. 9; Langston, p. 8; Clerf, p. 2; Helwig, p. 4; Macdonald, p. 14)

(37) The theory that lung cancer results from a direct-action contact carcinogen in tobacco smoke has long since been abandoned by most investigators. (Hockett, pp. 19-20)

(38) No ingredient or group of ingredients in cigarette smoke that is known to be responsible for human disease has been identified. (Hockett, p. 20)

(39) The SGAC Report, on p. 75, acknowledges that the amount of nicotine absorbed by smoking does not constitute any health problem. (Hockett, p. 15)